a finite number of agents utilizing a velocity alignment interaction and a Lennard-Jones potential, which provides both cohesive and repulsive interactions between neighboring agents. In the swarming regime of our model, an agent is selected at random to ''escape'' the flock, by choosing a particular direction to travel in, and no longer align with it's neighbors. We found that close to the swarming transition the escapee was unable to escape, while deeper in the swarming regime the swarm was more stable and the particle was able to escape with little effect on the rest of the swarm. Our research sheds light on the varied responses of swarms to internal dissent and suggests optimal strategies to escape or reorient swarms that exploit these responses.

2931-Pos Board B623

Post-Transcriptional and Post-Translational Control of the Flagellar Regulon Rescues Motility of a Salmonella Enterica Type III Export Flio Mutant

Clive S. Barker, Irina V. Meshcheryakova, Tomoharu Inoue, Fadel A. Samatey.
Trans-membrane Trafficking Unit, Okinawa Institute of Science and Technology, Okinawa, Japan.

The bacterial flagellum is made using a type III secretion pathway. For *Salmonella enterica*, this requires 6 transmembrane proteins FlhA, FlhB, FliO, FliP, FliQ, and FliR. FliO is not absolutely required, because bypass mutations in the *fliP* gene can improve motility for a *fliO* mutant. In this study, an extended screen was made for randomly selected mutations that improved motility of cells bearing a Δ*fliO* null mutation. Using whole genome sequencing two novel mutations were identified, a ''silent'' mutation localized to the *fliA* gene, which encoded a *U36C* substitution in the *fliAZ* mRNA. A missense mutation was found in the *clpP* gene, and encoded a V20F substitution in the ClpXP protease. Transcriptional and translational fusions of the *lacZ* gene to the *fliA* promoter demonstrated that the silent mutation improved translation of the highly structured *fliAZ* mRNA. Real-time quantitative RT-PCR revealed that late flagellar gene expression was blocked in a Δ*fliO* mutant, but the *clpP*(*V20F*) or *fliA*(mRNA *U36C*) mutations improved expression. Examination of flagellar biogenesis by immunoblotting and transmission electron microscopy found that the *fliO* mutant synthesized rare flagella, but introducing the *fliA*(mRNA *U36C*) mutation together with previously identified mutations in *fliP* cooperatively restored flagellar biogenesis to near wild-type levels.

2932-Pos Board B624

S. Aureus Adapt to Growth Conditions by Changing Membrane Order

Maria I. Perez[1], Steven M. Trier[2], Adriana Bernal[1], Juan Camilo Vargas[3], Cornelia Herrfurth[4], Ivo Feussner[4], John Mario Gonzalez[3], **Chad Leidy**[2].
[1]Biological Sciences, Universidad de los Andes, Bogota, Colombia, [2]Physics, Universidad de los Andes, Bogota, Colombia, [3]Medicine, Universidad de los Andes, Bogota, Colombia, [4]Plant Biochemistry, Georg-August-University, Goettingen, Germany.

Bacterial resilience has become a serious public health concern, as a population that can survive immunological and antibiotic attacks retains the ability to repopulate an infection. One mechanism by which bacteria may evade death is by changing the membrane physical properties, thereby providing defense against antimicrobials that act through membrane permeabilization. We hypothesize that the membrane properties are altered in different stages of bacterial growth and test this using S. aureus as a model system. Extracted lipids from the exponentially growing and attached biofilm cultures demonstrate similar phase behavior, whereas lipids from the stationary growth phase show a higher packing order at physiological temperatures, as measured using Laurdan generalized polarization. These results are confirmed in vivo by analyzing the spectral shift of Di-4-ANEPPDHQ alongside fluorescence anisotropy of DPH. Further analysis of the membrane components shows an increased expression of carotenoids in the stationary growth phase, and these carotenoids alter the phase behavior of synthetic lipid systems in a similar manner as measured in the bacterial lipid extracts. Oddly, changes in the pigmentation of biofilm cells with respect to the exponential growth phase are not noted, inferring that the collectively growing structures have sufficient nutrient perfusion, even in non-flow based culture models. We then test susceptibility to the membrane active peptides, Novocidin and Magainin, in purified bacterial membrane systems and live cells. This work suggests that bacteria alter their membranes in response to environmental stress and these adaptive changes provide increased resistance against natural membrane active antimicrobials.

2933-Pos Board B625

Long-Term Visualization of Micro Vortices in *Bacillus Subtilis* Bacterial Biofilm on Agar Plate using Particle Image Velocimetry

Kyunghoon Kim[1], Jung Kyung Kim[2,3].
[1]Department of Mechanical Engineering, Graduate School, Kookmin University, Seoul, Korea, Republic of, [2]School of Mechanical Systems Engineering, Kookmin University, Seoul, Korea, Republic of, [3]Department of Integrative Biomedical Science and Engineering, Graduate School, Kookmin University, Seoul, Korea, Republic of.

The collective dynamics of bacteria has a crucial role in the development of biofilm. Although the behavior of the fluidic biofilm was studied by many research groups, detailed mechanics of its motion still remains elusive. Previously, we developed a method for visualization of vortical flow in the *Bacillus Subtilis* (*B. subtilis*) colony using 200-nm fluorescent microbeads, which were initially embedded in the agar plate and distributed spontaneously at the upper surface of the growing colony. Here, we conducted a long-term live imaging of *B. subtilis* colony with the fluorescent beads and obtained high-resolution velocity maps of microscale vortices in the biofilm using particle image velocimetry (PIV). At the tip of the colony, a distinct periodic fluctuation of average speed and vorticity revealed by Micro-PIV analysis was correlated with switching between bacterial swarming and growth phases. Our advanced imaging tool helps to explain the effect of micro vorticies in the biofilm on the collective dynamics of bacteria.

2934-Pos Board B626

Non-Gauss Athermal Fluctuations in Bacterial Bath

Masato Aridome[1], Takashi Kurihara[1], Heev Ayade[1], Irwin Zaid[2], Daisuke Mizuno[1].
[1]Dept. Physics, Kyushu University, Fukuoka, Japan, [2]Oxford University, Oxford, United Kingdom.

Many active materials and biological systems are driven far from equilibrium by inclusions that spontaneously generate forces and give rise to flows/distortions in the surrounding material. Probing and characterizing these athermal fluctuations are essential to understand the properties and behaviors of such systems. Bacterial bath, where microscopic force generators (swimming E-coli) are randomly distributed in fluids, is a simple model system to study non-equilibrium fluctuation. We observe the trajectory of passive tracers dispersed in bacterial bath with video microscopy and analyze the lag-time dependent distribution of the displacements (van-Hove correlation function). It is found that for long lag times, the distributions are highly non-Gauss with broad tail, similar to those found in active cytoskeletal networks (actin gel actively actuated with myosin motor proteins). It has been found that the van Hove distribution in active cytoskeletal networks follows truncated Lévy statistics; since the fluctuation driven by single force generator presents power-law distribution with diverging variance, sum action of multiple motor proteins converges to Levy distribution rather than Gauss by generalized central limit theorem. However, the origin of non-Gauss fluctuations is still elusive in bacterial bath suspension.

Here we discuss an analogy between fluctuations in bacterial bath and those in active cytoskeletal networks, based on the fact that the impact of single force generator in these systems (velocity field around a bacteria and displacement field around a motor protein) both exhibit $1/r^2$ spatial decay.